Polychlorinated Biphenyl (PCB) and Dichlorodiphenyl Dichloroethylene (DDE) Concentrations in the Breast Milk of Women in Quebec

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ABSTRACT

Objectives. This study documented the concentration of polychlorinated biphenyls (PCBs) and dichlorodiphenyl dichloroethylene (DDE) in the breast milk of women from Quebec, Canada, and assessed the impact of various sociodemographic and lifestyle factors on these levels.

Methods. From 1988 to 1990, milk samples were obtained from 536 Quebec women and analyzed for seven PCB congeners and p,p'-DDE. Information was obtained on subjects' physical, sociodemographic, and lifestyle characteristics.

Results. Mean concentrations were 0.52 mg/kg lipids (95% confidence interval [CI] = 0.50, 0.54) and 0.34 mg/kg lipids (95% CI = 0.32, 0.35) for PCBs (Aroclor 1260) and DDE, respectively. Age and history of breast-feeding showed statistically significant correlations with PCB and DDE concentrations.

Conclusions. Concentrations of PCBs and DDE measured in this study are at the lower end of the concentration range recently reported for women living in industrialized countries. The modulating factors identified here should be considered when conducting studies on organochlorine exposure and disease. (*Am J Public Health.* 1996;86: 1241–1246)

Introduction

Polychlorinated biphenyls (PCBs) and dichlorodiphenyl trichloroethane (DDT) are persistent, lipophilic chlorinated organic compounds that have been used extensively in all parts of the world. PCBs have been used in heat exchange and dielectric fluid; as stabilizers in paints, polymers, and adhesives; and as lubricants in various industrial processes. DDT belongs to the chlorinated insecticide family and was used extensively from 1943 to 1972 for agricultural and public health purposes. In various organisms, DDT is slowly transformed to the even more stable and persistent DDE (dichlorodiphenyl dichloroethylene).[1]

In Canada as in most industrialized countries, PCBs are no longer being manufactured and are banned from use in new products and processes; for more than a decade now, their use has been restricted to closed systems in the electrical industry (transformers and condensers).[2,3] DDT was also banned in most industrialized countries during the 1970s. However, these chemicals are still being released into the environment because of inadvertent spills and careless disposal of PCBs and the ongoing use of DDT in developing countries for the control of insects that cause devastation to crops and human health.[4]

The high lipophilicity and the resistance to biodegradation of PCBs and DDE allow the bioaccumulation of these chemicals in fatty tissues of organisms and their biomagnification through food chains. These chemicals can be found in all ecosystems, including those located far away from industrialized regions, such as the Arctic aquatic ecosystem.[5] Because humans are located at the top of most food chains, relatively high levels of these compounds have been found in human adipose tissues, blood lipids, and breast milk fat. Fatty foods and fish are currently the major sources of exposure to PCBs and DDE in general populations from industrialized countries.[6,7]

Human milk monitoring programs have been implemented in various countries to assess the importance of human exposure to organochlorines and to follow temporal trends. Breast milk possesses a relatively high lipid content (3% to 4%), and levels of PCBs (Aroclor 1260) and DDE in the parts-per-million range (fat basis) have been documented in lactating women from various parts of the world.[3]

Organochlorine exposure has been the subject of renewed interest during the last 3 years, when several epidemiological studies reported an association between breast cancer risk and PCB or DDE concentration in mammary adipose tissues or plasma lipids.[8–10] Some PCB congeners and DDT isomers possess an endocrine-disrupting capacity, and their involvement in various reproductive and developmental disorders has been postulated.[11–13] To assess the magnitude of this potential public health problem correctly and, if needed, to adopt preventive measures, researchers must have body burden data in women of reproductive age and

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information regarding the modulating factors.

From 1988 to 1990, we conducted a large survey in the province of Quebec, Canada, to evaluate the exposure of the general female population to various organochlorine compounds. Levels of dioxinlike compounds and other chlorinated pesticides were determined in breast milk from a subsample of this population and were reported elsewhere.[5,14] Here we report on the levels of PCBs and DDE in breast milk samples from 536 women from the general Quebec population and on the main factors (sociodemographic, dietary habits, and lifestyle) associated with this contamination.

Methods

From December 1988 to May 1990, 536 women who had delivered in 22 randomly selected hospitals in nine administrative regions of Quebec province volunteered to participate in this study. Each region was represented by approximately 60 women. To be eligible, women were to have resided in their region for at least 1 year. Women who gave birth by cesarean section and women with multiple births were excluded from the survey.

For practical reasons and because of their specific dietary habits, populations inhabiting remote regions that rely on country food (fish, waterfowl, sea mammals) for subsistence—Arctic Quebec (north of 55°), the Cree region, and the Lower North Shore region along the St Lawrence River—were not included in this survey. A separate breast milk survey was conducted in Arctic Quebec during 1990 to 1991.[5]

Manual expression was used at home to collect milk samples (30 to 40 mL) between day 14 and day 21 after delivery. Collection duration did not exceed 24 hours. During this period, samples were kept in a 60-mL polycarbonate vial at 4°C in the home refrigerator. Once collection was completed, samples were frozen at −20°C in the home freezer. During the following days, a nurse from the local community health unit picked up the milk samples and sent them frozen to the laboratory for organochlorine analysis. The nurse also gathered information on breast milk collection and on the weight of the mother at the time of collection.

Concentrations of p,p'-DDE and of seven PCB congeners (IUPAC nos. 28, 52, 101, 138, 153, 180, and 183) in milk samples were determined by high-resolution gas chromatography. Milk samples were mixed with an aqueous solution of sodium oxalate, ethyl ether, and ethanol and then extracted with hexane. The hexane extracts were washed with reagent water and concentrated to constant weight, and the percentage of lipids was determined gravimetrically. The lipid residue was diluted in hexane and cleaned using a sulfuric acid–silica gel slurry, followed by elution through a neutral acid–silica gel chromatographic column. The analysis was performed by dual-column gas chromatography with a Hewlett-Packard (HP) 5890 gas chromatograph equipped with two capillary columns (HP Ultra 1 and Ultra 2, both 50-m long, 0.2-mm internal diameter, 0.33-μm coating). Twin electron capture detectors (HP) were used. The specific congeners were identified and quantified by comparing responses and retention times with calibration standards for each analyte of interest. The detection limit for DDE and for each PCB congener was 0.3 μg/kg lipids. To effect comparisons with results from previously published surveys, the total PCB (Aroclor 1260) concentration was calculated by multiplying by 5 the sum of the concentrations of congeners 138 and 153, since those congeners represent approximately 20% (by weight) of the Aroclor 1260 mixture.[15]

A pretested and self-administered bilingual questionnaire was used to collect information regarding age; weight before pregnancy, prior to delivery, and at 3 days postpartum; recent weight variations; alcohol consumption; location of dwelling; and number of previous children, as well as the date and duration of past breast-feeding experiences. The main part of the questionnaire was designed to assess animal fat and aquatic food consumption during the 12-month period prior to conception. Consumption of the principal species of fishes, both fresh and saltwater, was recorded according to Jacobson et al.[16] Questions pertaining to dietary fat consumption from meat and dairy products were adapted from those used during Canadian epidemiological studies on breast cancer.[17] The questionnaire was completed within 3 days of delivery at the hospital and was validated during a preliminary phase of this study among 20 women from both urban and rural settings.

For comparisons with data from other surveys, arithmetic means were used in the descriptive statistics. However, because contaminant concentrations follow a lognormal distribution, geometric means were used to create comparisons between subgroups, accompanied by the 95% confidence interval (reported below in brackets). Pearson correlation coefficients were used in the analyses based on quantitative variables. A multiple linear regression analysis was conducted to model the main factors found to be associated with milk contamination by univariate analysis. Correlation and regression analyses were performed with log-transformed values. All statistical analyses were conducted with the SAS package (SAS Institute Inc, Cary, NC).

Results

The participation rate varied from 75% to 95% in the 22 hospitals where the survey took place. The mean age of women was 27.9 years (SD = 4.2), ranging from 18 to 41 years. Most of the 536 women were multiparous (n = 293), and half (n = 267) were breast-feeding for the first time. Maternal mean body weight at the time of breast milk sampling (third week postpartum) was 63.0 kg (SD = 9.2), ranging from 42.7 to 97.7 kg. Mean birthweight of newborns was 3.43 kg (SD = 0.46), ranging from 1.93 to 4.97 kg.

PCB congeners 138, 153, and 180, as well as DDE, were detected in all samples, whereas PCB congeners 28, 52, 101, and 183 were detected in only 2.7%, 0%, 0.4%, and 4% of all samples, respectively. Therefore, the latter compounds were excluded from further statistical analysis. Table 1 presents the mean concentrations of PCBs and DDE in breast milk fat samples from the 536 women. DDE shows the highest concentration, followed by PCB congeners 153, 138, and 180. Frequency distributions for PCB (Aroclor 1260) and DDE concentrations exhibited lognormal characteristics, as generally observed in similar studies (data not shown). The Pearson correlation coefficient between DDE and PCB (Aroclor 1260) concentrations was 0.61 ($P < .0001$).

To eliminate the influence of past breast-feeding duration, the relationship between age and DDE and PCB (Aroclor 1260) milk concentrations was examined only in women who breast-fed for the first time. Breast-feeding lowers the body burden of these compounds, and its cumulative duration is expected to increase with age.[18] Age was positively related to both DDE and PCB milk concentrations. Mean levels of both organochlorines for women aged 30 years and more (PCBs = 0.65 mg/kg [0.60, 0.72]; DDE = 0.41 mg/kg [0.36, 0.47]) were greater ($P < .0001$) than those for

women aged less than 30 years (PCBs = 0.44 mg/kg [0.42, 0.48]; DDE = 0.27 mg/kg [0.25, 0.29]). With age as a continuous variable, Pearson coefficients were 0.44 ($P < .0001$) for PCBs and 0.46 for DDE ($P < .0001$).

All four weight variables (before pregnancy, prior to delivery, third day postpartum, and third week postpartum) showed small negative associations with PCB but not with DDE breast milk concentrations. The strongest correlation was observed with the third-week postpartum weight. For example, the mean concentration of PCBs for women weighing 62 kg or more at this period was smaller than that for women weighing 61 kg or less (0.44 mg/kg [0.42, 0.47] vs 0.50 mg/kg [0.47, 0.53]; $P < .005$). The Pearson r coefficient for the relationship with PCBs, using weight at third week postpartum as a continuous variable, was $-.17$ ($P < .0001$). A slightly improved correlation coefficient was obtained when the body mass index (weight/height^2), an index of obesity, was used as the independent variable ($r = -.22$; $P < .0001$). Recent weight loss (occurring between delivery and milk collection) and maternal height were not associated with organochlorine concentration.

Investigation of the relationship between PCB and DDE concentration in breast milk and breast-feeding history revealed that mean concentrations decreased regularly with breast-feeding duration. Age-adjusted mean levels of both organochlorines for women who never breast-fed (PCBs = 0.52 mg/kg [0.50, 0.55]; DDE = 0.33 mg/kg [0.31, 0.35]) were greater ($P < .0001$) than those for women who ever breast-fed (PCBs = 0.41 mg/kg [0.39, 0.43]; DDE = 0.25 mg/kg [0.24, 0.27]). When the cumulative breast-feeding duration was entered as a continuous variable, age-adjusted partial correlation coefficients were $-.31$ ($P < .0001$) for DDE and $-.33$ ($P < .0001$) for PCBs.

To separate the influence of parity from that of the duration of lactation, the age-adjusted concentrations of PCBs and DDE were compared in primiparous women, multiparous women who never breast-fed, and multiparous women who ever breast-fed (Table 2). Statistically significant differences were observed between primiparous and multiparous women with past breast-feeding experience ($P < .0001$) but not between primiparous and multiparous women who never breast-fed ($P > .1$).

Concentrations of both DDE and PCBs increased with total fish consump-

TABLE 1—Concentration of PCBs and DDE in Breast Milk Fat from 536 Quebec Women

Organochlorine	Arithmetic Mean, mg/kg Lipids	95% CI	Range
DDE	0.34	0.32, 0.35	0.02–2.89
PCB congener 138	0.046	0.044, 0.048	0.01–0.18
PCB congener 153	0.054	0.052, 0.057	0.01–0.20
PCB congener 180	0.027	0.026, 0.027	0.01–0.09
Total PCB (Aroclor 1260)	0.52	0.50, 0.54	0.07–1.88

Note. CI = confidence interval; DDE = dichlorodiphenyl dichloroethylene; PCBs = polychlorinated biphenyls.

TABLE 2—Age-Adjusted Concentration of PCBs and DDE in Breast Milk Fat, by Parity and Breast-Feeding Experience

Parity/Breast-Feeding	Organochlorine	Geometric Mean, mg/kg Lipids	95% CI
Primiparous (n = 242)	PCBs	0.53	0.50, 0.56
	DDE	0.33	0.31, 0.35
Multiparous/never breast-fed (n = 25)	PCBs	0.47	0.40, 0.56
	DDE	0.31	0.25, 0.37
Multiparous/ever breast-fed (n = 268)	PCBs	0.41	0.40, 0.43
	DDE	0.25	0.24, 0.27

Note. CI = confidence interval; DDE = dichlorodiphenyl dichloroethylene; PCBs = polychlorinated biphenyls.

tion. The mean concentration of PCBs for women who never ate fish was smaller than that for women who ate more than one fish meal per week (0.35 mg/kg [0.29, 0.42] vs 0.47 mg/kg [0.45, 0.49]; $P < .001$). A similar difference was observed for DDE levels between the two groups (0.23 mg/kg [0.19, 0.28] vs 0.29 mg/kg [0.28, 0.30]; $P = .03$). The Pearson r correlation coefficients were .12 ($P = .004$) for DDE and .16 ($P = .0002$) for PCBs when fish consumption was entered as a continuous variable. Fat consumption from meat and dairy products was not associated with organochlorine concentration in breast milk.

Alcohol consumption was positively related to both DDE and PCB concentrations in breast milk. The mean concentration of PCBs for women who never consumed alcohol was smaller than that for drinkers (0.38 mg/kg [0.33, 0.44] vs 0.47 mg/kg [0.46, 0.50]; $P = .0001$). A similar difference was noted for DDE concentrations between the two groups (0.23 mg/kg [0.20, 0.26] vs 0.29 mg/kg [0.28, 0.31]; $P = .002$). Statistically significant Pearson r coefficients were obtained for both organochlorines when alcohol

consumption was entered as a continuous variable (PCBs: $r = .21$, $P < .001$; DDE: $r = .16$, $P < .0001$).

A multiple linear regression analysis showed age, duration of past breast-feeding episodes, fish consumption, third-week postpartum weight, and alcohol consumption to have statistically significant relationships with PCB breast milk concentration (Table 3). The model explained 24.9% ($P < .0001$) of PCB concentration variation in breast milk. When the analysis was restricted to women who never breast-fed, this percentage reached 28.9% ($P < .0001$). For DDE (all women included), only age and duration of breast-feeding displayed a statistically significant association with DDE concentration (multiple linear regression; $r^2 = .21$; $P < 0.001$) (Table 4).

Discussion

Monitoring of breast milk concentration may be used to assess temporal and geographical variations in human exposure to lipophilic and persistent xenobiotics.[3] To ensure comparability with other surveys, milk samples were collected

TABLE 3—Multiple Regression Analysis of Polychlorinated Biphenyl (PCB) (Aroclor 1260) Concentration in Breast Milk Fat

Source	df	Mean Square	F	P
Age	1	3.04	93.69	.0001
Weight	1	0.45	13.92	.0002
Cumulative breast-feeding duration	1	1.51	46.65	.0001
Fish consumption	1	0.18	5.67	.0176
Alcohol consumption	1	0.21	6.45	.0114

TABLE 4—Multiple Regression Analysis of Dichlorodiphenyl Dichloroethylene (DDE) Concentration in Breast Milk Fat

Source	df	Mean Square	F	P
Age	1	5.94	126.50	.0001
Cumulative breast-feeding duration	1	2.78	59.06	.0001

during the third week of lactation, according to the World Health Organization procedure.[19] This period of lactation is too short to allow for a significant excretion of organochlorine, so levels observed at this time are representative of the maternal body burden.

Mean breast milk concentrations of DDE and PCBs were reported in several parts of the world from 1960 to 1991.[3] A downward trend has been observed in some but not all industrialized countries. In Sweden (Stockholm), PCB (Clophen A 50) concentrations in milk fat were 0.93 mg/kg in 1976/77, 0.88 mg/kg in 1978, 0.79 mg/kg in 1979, 0.76 in 1980, and 0.60 in 1985; since 1985, these have stabilized at about 0.65 mg/kg.[3] In Canada, mean DDE concentrations in milk fat declined from 3.84 mg/kg[20] in 1967/68 to 0.91 mg/kg in 1982,[21] and to 0.33 mg/kg in 1986.[22]

In a previous study conducted during 1978 to 1979 in the province of Quebec, 154 breast milk samples (immature milk) were collected and analyzed for PCBs (Aroclor 1254) and DDE; the respective mean concentrations of these organochlorines in milk fat were 0.84 (SD = 0.53) and 0.88 (SD = 0.87) mg/kg.[23] DDE levels reported in the present study (mean = 0.34 mg/kg; SD = 0.21) indicate a 61% decrease when compared with data obtained 10 years ago.

Present levels of DDE in Quebec are in the lower range of levels reported recently in industrialized countries. For example, the mean concentration observed in our study for the period 1988 to 1990 was 0.34 mg/kg, compared with 0.35 mg/kg in Sweden during 1990,[24] 0.53 in Germany during 1990,[25] 0.60 in Spain during 1991,[26] and 2.0 in New Zealand during 1987/88.[27] This conclusion holds true for the remote Arctic regions of Quebec, where Inuit women displayed higher levels of DDE than southern Quebec women. Results of 107 breast milk analyses conducted in 1989/90 showed a mean DDE concentration of 1.2 mg/kg lipids, a level five times greater than that measured in breast milk samples of women living in the southern part of the province.[5]

Comparison with other studies regarding PCB levels is difficult because of variations in methods used for quantification (number of peaks analyzed, PCB standard). Here we calculated the PCB (Aroclor 1260) concentration by multiplying the sum of PCB congener 138 and 153 concentrations by 5, yielding a concentration of 0.52 mg/kg. Using the congener-specific data reported by Mes and colleagues,[28] we similarly calculated the mean PCB (Aroclor 1260) concentration in breast milk fat from Canadian women (all meridional regions) to be 0.93 mg/kg in 1982 and 0.32 mg/kg in 1986. Mean PCB (Aroclor 1260) concentrations of 2.3, 1.6, 1.0, and 0.36 mg/kg were calculated using congener-specific data for milk samples collected in Germany during 1990,[25] in the Netherlands from 1990 to 1992,[29] in Norway during 1991,[30] and in New Zealand during 1987/88,[27] respectively. In Arctic Quebec, results from the 1989/90 survey revealed a mean concentration of 2.9 mg/kg, a level five times

greater than that of southern Quebec women in the present survey.[31]

Several factors have been linked to the levels of organochlorines in breast milk. First, since lactation is the most important route of excretion for persistent organochlorines,[18] the negative relationship observed between total duration of cumulative past breast-feeding duration and DDE or PCB concentration was expected. Most previous studies reported that organochlorine levels in milk fat or whole milk decrease significantly with parity, a surrogate for past breast-feeding duration. Failure to adjust for age, which is related to both parity and organochlorine concentration in breast milk, may explain why some researchers did not observe this association.[18] Results presented in Table 2 indicate that lactation and not parity displays a negative relationship with concentrations of DDE and PCBs in breast milk fat.

Concentrations of PCB congeners 138, 153, and 180 and DDE in body fat are expected to increase with age because the biological half-lives of these chemicals are very long.[32,33] This has been repeatedly observed in population surveys using blood samples or adipose tissue samples.[34,35] However, breast milk studies have reported contradictory results. While some investigators observed lower PCB concentrations in young women,[36,37] Polishuk et al.[38] noted an inverse relationship and others[39–41] did not report any association between organochlorines and age. These discrepancies may be due to the small sample size of most studies and the narrow age range of women during their reproductive years. The large sample size in our study (n = 536) allowed us to observe the expected accumulation of organochlorines with age. Furthermore, this relationship can be obscured by not controlling for cumulative past breast-feeding duration, which likely increases with age.

Only PCB levels showed a statistically significant association with body weight in the present study. Since DDE and PCBs are stored in body fat, an inverse relationship between body weight and levels of these organochlorines in breast milk fat may be expected. This inverse relationship has been reported in most studies to date,[18] but some researchers have not observed it.[42,43] In many instances, body weight may not be a valid surrogate of body fat weight; in our study, a slightly higher correlation was observed using body mass index, an index of obesity, instead of body weight.

Several studies have indicated that eating contaminated fish and marine products may be the most important dietary factor influencing the concentrations of DDT and PCBs in human milk.[37,40,44–48] The present results also indicate an association between fish consumption and DDE and PCB milk-fat level. This relationship is even more striking for populations that depend on sea products for subsistence, such as the Inuit of Arctic Quebec[5,31] and the fishermen from the Lower North Shore region of the St. Lawrence River.[49]

Alcohol consumption was associated with both DDE and PCB concentrations in breast milk fat. This association was reported previously[34,50] although the reason for the relationship is not clear. Alcohol abuse can induce chronic liver damage, which may in turn reduce the biotransformation rate of these chemicals.[51] Alternatively, alcohol could increase absorption of organochlorines by raising the solubility of DDE and PCBs in gastrointestinal fluids. Indeed, ethanol is an excellent solvent for many drugs and is frequently used as a vehicle for medicinal mixtures.[52]

Multiple linear regression models constructed to explain the variation of PCB and DDE concentrations in breast milk, while statistically significant, were successful in explaining less than half of the total variance. Inclusion of information regarding direct exposure to PCBs in chemical plants or electrical industries, and to DDT during farming or gardening activities, would have enhanced the validity of the model. In addition, host variables affecting biotransformation, such as genetic variation, enzyme induction, nutritional status, and hepatic injury,[51] were not considered in the model but may modulate organochlorine levels in breast milk. For example, Kreiss and coworkers[34] reported that adults who were exposed to DDT/DDE through fish consumption and took liver enzyme–inducing medications (hydantoin, barbiturates, major tranquilizers) in the year before the cross-sectional study had substantially lower DDT/DDE levels than did DDT/DDE-exposed adults who did not take these medications.

No adverse health effect is expected in the general adult population that was exposed to relatively low environmental levels of PCBs similar to those encountered in the present study.[53,54] However, Colborn and colleagues[11] suggest that adverse effects on reproduction and devel-opment, as well as carcinogenic effects, may be occurring in human populations as a result of environmental exposure to organochlorines, some of which have been described as endocrine-disrupting chemicals. In particular, several recent epidemiological studies reported an association between breast cancer risk and PCB or DDE concentration in mammary adipose tissues or plasma lipids.[8–10] Case–control studies are currently under way to substantiate this relationship further. If meaningful conclusions are to be reached, these studies will have to control for confounding factors such as age, weight, and history of breast-feeding, which have been shown in this paper and by others[18] to influence the organochlorine body burden, as well as the breast cancer risk,[55,56] of women throughout their lives. □

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